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Waist to Hip Ratio versus BMI as Predictor of Obesity Related Pregnancy Outcomes

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Abstract

Objective—In nonpregnant populations the waist to hip ratio (WHR) is a better predictor of obesity related outcomes than BMI. Our objective was to determine, in pregnancy, the relationship between these measures of obesity and LGA and cesarean delivery (CD).

Methods—This is a secondary analysis of data from the Combined Antioxidant and Preeclampsia Prediction Study (CAPPS). Women a WHR of ≥ 0.85 and 0.80 – 0.84 at 9–16 weeks gestation, were compared to those with a WHR <0.80 . Women with early pregnancy BMI ≥ 30.0 kg/m² (obese) and 25.0 – 29.9 kg/m² (overweight) were compared to those <25.0 kg/m². LGA was defined as $>90\%$ by Alexander nomogram. Univariable analysis, logistic regression and ROC curves were used.

Results—Data from 2,276 women were analyzed. After correcting for potential confounders, only BMI ≥ 30 was significantly associated with LGA (aOR 2.07, 1.35–3.16) while BMI 25.0 – 29.9 (aOR 1.5, 0.98–2.28), WHR 0.8 – 0.84 (aOR 1.33, 0.83–2.13) and WHR ≥ 0.85 , (aOR 1.05, 0.67–1.65) were not. Risk for CD was increased for women with elevated WHR and with higher BMI compared to normal.

Conclusion—WHR is not associated with LGA. While BMI performed better than WHR, neither was a strong predictor of LGA or need for CD in low risk nulliparous women.

Introduction

Obesity is now considered to have reached epidemic proportions. During pregnancy, obesity has been associated with increased maternal and neonatal morbidity which includes an increased risk for large for gestational age infants (LGA).¹ LGA fetuses are more likely to experience stillbirth, shoulder dystocia and admission to the neonatal intensive care unit compared to normal weight fetuses.^{2,3} Multiple prior studies have linked LGA to increasing maternal body mass index (BMI).^{4–8} A study of singleton births found the strongest modifiable predictor of LGA to be prepregnancy weight.⁹ The outcome is highly multifactorial as other studies link parity, gender, race, glucose intolerance, diabetes, and maternal weight gain to LGA.^{10–12}

BMI is an estimate of body fatness used as a screening tool for obesity and predictor of obesity related disorders.¹³ The correlation of BMI and body fatness varies with age, race, gender and athletic ability and does not have the ability to differentiate fatness as central or visceral. Alternatively, as a screening tool, the waist to hip ratio (WHR) has been shown to be more reflective of visceral fat and central adiposity, as well as a better predictor of obesity related disorders, such as stroke, myocardial infarction, or cardiovascular death, than BMI.^{14–19} This anthropometric index has not been as widely studied in relation to pregnancy outcomes, but in a prospective cohort of pregnancy, both increased WHR and BMI were associated with an increased risk of preeclampsia.²⁰ Central adiposity and LGA infants are both associated with elevated triglycerides, LDL cholesterol, free fatty acids, fasting glucose and insulin, suggesting fat distribution may affect fetal growth independent of fat mass.^{21–23} In addition, central adiposity is associated with variations in the concentrations of specific sex hormones which may play a role in the maternal regulation of fetal growth.²⁴ The WHR is largely unaffected until 20 weeks of gestation.^{21,24} A measurement in early pregnancy would be a reliable indicator of central adiposity.

Our objective was to determine the relationship between WHR in early pregnancy and LGA and CD, as well as compare the predictive value of this measure of obesity to BMI in a population of low risk nulliparous women.

Methods

This is a secondary analysis of the Combined Antioxidant and Preeclampsia Prediction Study (CAPPS), a multicenter randomized control trial of vitamin C and E to prevent preeclampsia which enrolled 9,969 nulliparous women prior to 16 weeks gestation.²⁵ For the secondary analysis inclusion criteria included women with data available to determine WHR and BMI at the time of enrollment. Exclusion criteria included congenital anomalies and intrauterine fetal demise. Intervention and control groups were combined into one cohort for analysis. WHR was evaluated as a categorical variable, comparing women with a ratios of 0.85, 0.80–0.84, and <0.80 representing high, moderate and low risk respectively for adverse outcomes.¹⁶ Likewise, BMI was also evaluated categorically, comparing 30 kg/m² (obese) and 25–29 kg/m² (overweight) to <25 kg/m². Outcomes were also measured using both WHR and BMI as continuous variables.

The primary outcome was LGA as defined by birth weight >90% by the Alexander nomogram.²⁶ Secondary outcomes included overall need for cesarean delivery (CD) and need for CD due to cephalopelvic disproportion. Univariable analysis was performed followed by multivariable logistic regression adjusting for maternal age, gestational age at enrollment, years of schooling, race, alcohol, and smoking status. The Breslow-Day test for homogeneity was used to determine if there was a difference in the association of WHR and BMI with LGA and CD by race/ethnicity. Receiver operator curves (ROC) were calculated to determine the relationship between both WHR and BMI with the incidence of LGA and need for CD. Categorical variables were analyzed using the chi square test or Fisher's exact test. Continuous variables were analyzed using the Wilcoxon rank sum test or the Kruskal-Wallis test. Statistical analysis was conducted with SAS software (SAS Institute, Cary, NC). A nominal two-sided P value less than 0.05 was considered to indicate statistical significance and no adjustments were made for multiple comparisons.

Results

Of the 9,969 women analyzed in the CAPPS trial, 2,276 met inclusion criteria and had data available for calculation of WHR and BMI at < 16 weeks gestation. Group demographic information is displayed in Table 1.

On initial univariate analysis, WHR was not significantly associated with LGA. BMI was associated with LGA. Both increased WHR and BMI were associated with an increased need for CD.

Results of the multivariable regression are displayed in Table 2. BMI ≥ 30 remained significantly associated with LGA, but WHR was not. Both categories of elevated WHR and BMI remained predictive of CD. There were no significant interactions between race/ethnicity and WHR or BMI for these outcomes (data not shown).

ROC curves for the risk of LGA and CD are displayed in Figures 1 and 2. WHR performed no better than chance alone in prediction of LGA (AUC 0.514, $p=0.5724$) while increased BMI was slightly more predictive of LGA (AUC 0.615, $p<0.001$). Both variables were similarly associated with CD and both significantly more predictive than chance alone (WHR AUC 0.58, $p<0.0001$; BMI AUC 0.61, $p<0.0001$). These curves were similar when only CD performed for cephalopelvic disproportion was analyzed.

Data were then further analyzed by comparing outcomes from women meeting both criteria of WHR ≥ 0.85 and BMI ≥ 30 kg/m² to those meeting only one criteria or neither criterion. On univariate analysis, there was a significant difference between groups for both prediction of LGA and CD (Table 3). After multivariable regression, the OR of LGA for women with both WHR ≥ 0.85 and BMI ≥ 30 kg/m² was no longer significant as compared to nonobese women (OR 1.40, 95% CI 0.86–2.29), but the risk of CD was significantly elevated (OR 2.48, 95% CI 1.88–3.28).

Discussion

In this cohort of low risk nulliparous women, we found no association between increased WHR and the incidence of LGA, while maternal obesity (BMI ≥ 30) in early pregnancy was predictive of development of LGA. Both measurements were similarly predictive of the need for CD.

Multiple prior studies have found similar results in regards to the relationship between BMI and LGA or macrosomia.⁴⁻⁷ A prospective cohort study in Sweden found women with a BMI of 35.1–40 and a BMI of >40 have an adjusted odds ratio of 3.11 and 3.82 respectively for having an LGA infant.¹⁰ These findings are supported by other studies noting increased risks for LGA infants when mothers were obese (OR ranges 2.32–3.1).^{7,8}

Three previous studies have evaluated the relationship between WHR and infant size. Brown et al prospectively followed 521 low risk, primarily Caucasian women with singleton pregnancies who had WHR measured in early pregnancy. They found that the birthweight increased by 120 gm for each 0.1 unit increase in WHR, but did not specifically address birthweight percentiles.²⁴ A separate study of 700 Caucasian women, found central obesity, as defined by WHR of >0.9 , to have a 1.81-fold increased risk of LGA.²⁷ Salem et al, in a cohort study of nulliparous women in the United Kingdom, noted a WHR in the third and fourth quartiles of their cohort was associated with macrosomia, as defined by birthweight ≥ 4000 g, or LGA, as defined by birthweight $\geq 95\%$ of the cohort.²¹ The median values of the third and fourth quartile for WHR were 0.75 and 0.81 respectively, lower than in our US-based study. A further study evaluating the association of waist circumference with macrosomia in Brazilian women found an AUC for the ROC curve to be 0.645, similar to the predictive value of BMI, which had an AUC of 0.632.²⁸ The variations in study design and differences in patient population may explain these differences. In their study, Brown et al initially controlled for gestational diabetes, but found that the variable did not contribute to outcomes and subsequently dropped it from further analysis.²⁴ Bo et al also controlled for the presence of gestational diabetes in their study and found the increased rate of LGA to be independent of its presence.²⁷ As the predictive value of elevated WHR was being evaluated in our study, the incidence of GDM and gestational weight gain were not included in our multivariable analysis, given these variables lie in the causative pathway for the studied outcomes. In addition, obesity itself has been shown to be independently associated with infant size.^{5,29}

Central adiposity is associated with the presence of visceral adipose tissue, which is associated with multiple abnormal markers of metabolism including insulin resistance, dyslipidemia, and hypertension.¹⁷ In turn, these markers have been shown to be predictive of fetal size. While an excess of glucose supply may cause fetal hyperinsulinemia and lead to macrosomia, other abnormal metabolic factors may also contribute to fetal growth.^{29,30} Studies of women with positive diabetic screens but normal glucose tolerance tests found fasting maternal hypertriglyceridemia in the third trimester to be predictive of LGA infants independent of prepregnancy BMI.^{23,30} Other studies have found an association between maternal free fatty acid concentrations or amino acids with fetal weight.^{23,29} These abnormalities are hypothesized to lead to increased fetal growth as the hydrolysis of

maternal triglycerides by placental lipoprotein lipase leads to increased free fatty acids crossing the placenta.²³ We did not find the hypothesized association between central adiposity and fetal size, but this is possibly due to the low risk nature of the cohort.

Our finding that the rate of CD increases with BMI is consistent with prior studies.^{4,5,7,8,31} In cohort studies focused on singleton pregnancies, odds ratios for CD of 1.35 for overweight women to 2.92 for morbidly obese women (BMI>40) were found compared to women with normal BMI.^{7,8,10} This outcome was not reported by Brown, and Salem found no difference in the rate of spontaneous birth between quartiles, but used a lower cutoff value for WHR.^{21,24} In their cohort, Bo et al found women with a WHR of >0.9 had a 1.51-fold increased risk of CD, similar to our results.²⁷ Although we did not find an increased incidence of LGA associated with increased WHR, the abnormal markers of metabolism associated with both central adiposity and abnormal fetal growth may affect other measures of newborn size which may then contribute to cephalopelvic disproportion. For example, Brown et al found a 0.3 cm increase in head circumference for every 0.1 increase in WHR.²⁴ Further study would be required to determine if other neonatal anthropometric measurements are affected by maternal central adiposity.

Weaknesses of this study include the fact that it is a secondary analysis and therefore data is limited by availability from the primary study. We did not have information available for other risks associated with LGA, such as shoulder dystocia, obstetrical laceration, or neonatal hypoglycemia. Strengths included that the study population is a large, heterogeneous group from multiple institutions across the United States, and therefore applicable to wider populations than prior studies. The fact that only nulliparous patients were included eliminates further bias as WHR increases with parity.

In conclusion, in a pregnant heterogeneous low risk cohort, BMI but not WHR is predictive of LGA. Both measurements are similarly predictive of the need for CD, and further study is required to determine if this is due to an alteration in neonatal growth or other factors related to obesity.

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Figure 1.
ROC curve of LGA

Figure 2.
ROC curve of CD

Table 1

Group demographics by WHR and BMI

	WHR <0.8 n=639	WHR 0.8–<0.85 n=588	WHR 0.85 n=1049	P value	BMI <25.0 kg/m ² n=1184	BMI 25.0 – 29.9 kg/m ² n=597	BMI 30.0 kg/m ² n=495	P value
Age	23.5 ± 4.9	23.8 ± 4.8	23.1 ± 4.7	0.01	23.5 ± 4.8	23.2 ± 4.8	23.2 ± 4.6	0.17
Gestational age at enrollment	11.4 ± 1.1	11.5 ± 1.1	11.4 ± 1.1	0.30	11.5 ± 1.1	11.4 ± 1.1	11.3 ± 1.1	0.004
Years of schooling	13.6 ± 2.2	13.5 ± 2.3	12.3 ± 2.7	<0.001	13.2 ± 2.7	12.8 ± 2.7	12.7 ± 2.2	0.001
Race				<0.001				<0.001
Hispanic	60 (9.4%)	106 (18.0%)	416 (39.7%)		300 (25.3%)	181 (30.3%)	101 (20.4%)	
AA	213 (33.3%)	125 (21.3%)	207 (19.7%)		212 (17.9%)	138 (23.1%)	195 (39.4%)	
White	354 (55.4%)	342 (58.2%)	408 (38.9%)		644 (54.4%)	268 (44.9%)	192 (38.8%)	
Other	12 (1.9%)	15 (2.6%)	18 (1.7%)		28 (2.4%)	10 (1.7%)	7 (1.4%)	
Alcohol use	76 (11.9%)	66 (11.2%)	102 (9.7%)	0.34	129 (10.9%)	54 (9.1%)	61 (12.3%)	0.21
Smoking	90 (14.1%)	97 (16.5%)	188 (17.9%)	0.12	170 (14.4%)	105 (17.6%)	100 (20.2%)	0.009

Table 2
Rates and Adjusted Odd Ratios (aOR) for LGA and Cesarean Delivery by WHR and BMI Categories

WHR	No./Total (%)	aOR (95% CI)	BMI (kg/m ²)	No./Total (%)	aOR (95% CI)
LGA					
<0.80	35/639 (5.5)	Referent	<25.0	54/1182 (4.6)	Referent
0.80–84	42/587 (7.2)	1.33 (0.83, 2.13)	25.0–29.9	41/597 (6.9)	1.50 (0.98, 2.28)
0.85	64/1048 (6.1)	1.05 (0.67, 1.65)	30.0	46/495 (9.3)	2.07 (1.35, 3.16)
CD					
<0.80	116/639 (18.2)	Referent	<25.0	225/1184 (19.0)	Referent
0.80–84	142/588 (24.1)	1.43 (1.08, 1.89)	25.0–29.9	153/597 (25.6)	1.45 (1.14, 1.84)
0.85	303/1049 (28.9)	1.74 (1.35, 2.25)	30.0	183/495 (37.0)	2.52 (1.98, 3.21)

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Table 3

Rates for LGA and Cesarean Delivery by One or Both Obesity Categories

	Not Obese	BMI 30 kg/m ²	WHR 0.85	Both Obesity Categories	P value
LGA No./Total (%)	58/1061 (5.5)	19/165 (11.5)	37/718 (5.2)	27/330 (8.2)	0.006
CD No./Total (%)	200/1062 (18.8)	58/165 (35.2)	178/719 (24.8)	125/330 (37.9)	<0.001